ENVIRONMENTAL TOBACCO SMOKE

CAS Registry Number: N/A

Molecular Formula: N/A

Environmental tobacco smoke (ETS) is the smoke from tobacco products to which non-smokers are exposed. ETS is comprised of exhaled mainstream smoke and sidestream smoke. Exhaled mainstream smoke consists of smoke exhaled by the smoker. Sidestream smoke is the material emitted from smoldering tobacco products between puffs. A small amount of smoke is also emitted at the mouthpiece during puff drawing and, for cigarettes and cigars, by diffusion through the wrapper (NRC, 1986 as reported in U.S. EPA, 1992b).

The same chemical constituents are present in mainstream smoke and sidestream smoke. The quantitative differences in the individual constituents in mainstream and sidestream smoke emissions are the result of important physical and chemical differences in the burning conditions during their generation. Consequently, many constituents have a higher rate of release into sidestream smoke than mainstream smoke (U.S. EPA, 1992b). Table I presents some of the more common components of ETS.

SOURCES AND EMISSIONS

A. Sources

ETS is emitted from the combustion of tobacco or tobacco-containing products such as cigars, pipes, and cigarettes, with cigarettes being the most common product form. These emissions contain both vapor-phase and particulate contaminants. Sidestream smoke is the major component of ETS, contributing to nearly all of the vapor-phase constituents and over half of the particulate matter (U.S. EPA, 1992b).

B. Emissions

Researchers have identified over 4,000 individual constituents in ETS, many of which are known or suspected human carcinogens and toxic agents. Benzene, 1,3-butadiene, polycyclic aromatic hydrocarbons, N-nitrosamines, nicotine, and particulate matter are just some the toxic chemicals released during the burning of tobacco products (U.S. EPA, 1992b).

The California Air Resources Board (ARB) sponsored a study that provides emission factors for selected volatile organic compounds, $PM_{2.5}$, and other toxic air contaminants in ETS. Emission data from that study are presented in Table II. The presented data are the average

emissions from six commercial cigarettes representing brands that are widely smoked in California. Data are also presented for a Kentucky reference cigarette. This is the only study of ETS emissions performed specifically on the most popular brands of cigarettes in California and adds to the limited ETS data previously available (Daisey et al., 1994).

C. Natural Occurrence

ETS is a by-product of tobacco smoking and does not have any natural sources.

AMBIENT CONCENTRATIONS

ETS dilutes with ambient air. Its constituent gases and particulate matter mix with background concentrations and are then detectable only as individual compounds. Many of these compounds (e.g. benzene) are routinely monitored in the ambient air by ARB and ambient concentrations of specific compounds are included in those compounds' summary sheets.

Using *iso*- and *anteiso*-alkane concentration patterns, Rogge (1993) estimated that the contribution of cigarette smoke to ambient fine particulate matter in the Los Angeles area averaged approximately 0.57 to 0.72 micrograms per cubic meter ($\mu g/m^3$) during 1982.

INDOOR SOURCES AND CONCENTRATIONS

ETS is a major contributor to indoor air pollution whenever smoking occurs.

The measured concentrations of individual ETS constituents in indoor environments show marked spatial and temporal variations. Factors affecting indoor concentrations include the generation rate of the constituents from the tobacco product, the location at which the smoking occurs, the rate of tobacco consumption, the ventilation or infiltration rate, air mixing, the removal of contaminants, and the re-emission of contaminants from surfaces (U.S. EPA, 1992b).

While a number of compounds have been used to represent ETS concentrations in indoor environments, vapor-phase nicotine and respirable suspended particulates (RSP) have been the most widely used markers for the presence and concentration of ETS. However, because even these constituents have drawbacks, researchers are continuing to look for better markers for ETS. Measured concentrations of nicotine in indoor residential environments typically range from 2 to 10 $\mu g/m^3$ but may exceed 70 $\mu g/m^3$ in restaurants and in transportation vehicles (U.S. EPA, 1992b). RSP (fine particles that are able to infiltrate deep into the human lung) concentrations, similar to the findings of nicotine, vary widely in indoor environments. In residential environments, smoking has been estimated to increase RSP levels from 18 to 95 $\mu g/m^3$ with one individual increase measured at 560 $\mu g/m^3$ (background levels were subtracted out). Increases in restaurants may exceed 1,000 $\mu g/m^3$ (U.S. EPA, 1992b).

In California homes with smokers, the mean measured PM_{10} (respirable particles which have diameters less than 10 microns) concentrations were 125.6 $\mu g/m^3$ for a 12-hour daytime

measurement and 92.9 $\mu g/m^3$ for a 12-hour nighttime measurement. In contrast, in homes where no smoking occurred, daytime PM_{10} levels averaged 87.8 $\mu g/m^3$ and nighttime levels averaged 54.6 $\mu g/m^3$ (Pellizzari et al, 1992). In addition, in many field studies, specific toxic constituents of ETS such as benzene, 1,3-butadiene, and polycyclic aromatic hydrocarbons have been measured at much higher levels in homes where smoking occurred than in homes where no smoking occurred.

ATMOSPHERIC PERSISTENCE

ETS is a complex mixture of chemicals in the gas and particle phase, with a number of compounds being distributed between the gas and particle phases (Eatough et al., 1989; Benner et al., 1989). The gaseous chemicals are subject to gas phase reactions with hydroxyl radicals, NO_3 radicals, ozone, and gaseous nitric acid, as well as photolysis, while the particles and particle-associated chemicals are subject to wet and dry deposition (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics "Hot Spots" Program (AB 2588). Of the risk assessments reviewed as of December 1996, ETS was not listed in any of the risk assessments (OEHHA, 1996a,b).

HEALTH EFFECTS

The most probable route of human exposure to ETS is inhalation.

Non-Cancer: Children exposed to ETS from parental smoking have been found to have increased respiratory irritation, middle ear effusion, and a small reduction in lung function. Exposure of young children and particularly infants to ETS may cause an increased risk of pneumonia, bronchitis, and bronchiolitis. Exposure to ETS causes additional episodes and increased severity of asthma attacks. It is suspected that passive smoke may cause asthma in previously asymptomatic children. Passive smoking causes coughing, phlegm production, chest discomfort and reduced lung function in exposed adults (U.S. EPA, 1992b).

The State of California under Proposition 65 has determined that smoking (tobacco smoke, primary) causes developmental toxicity (CCR, 1996). Environmental tobacco smoke is not currently listed by the State of California under Proposition 65 as a reproductive or developmental toxicant. Everson et al. (1986) have reported an association between ETS exposure (spousal smoking) and a lower age at menopause. Haddow et al. (1988) reported that ETS exposure of pregnant women, as reflected in blood cotinine concentrations, was associated with a decrement in birthweight. An association has also been reported between infant exposure to environmental tobacco smoke and the incidence of Sudden Infant Death Syndrome (SIDS) (Klonoff-Cohen et al., 1995). In children whose mothers smoked, investigators reported a dose-related effect on scores from a Behavior Problem Index completed by the mothers when the children were 4 to 11 years old (Weitzman et al., 1992).

Cancer: Results from epidemiological studies indicate that ETS causes lung cancer in adults (U.S. EPA, 1992b). ETS has also been causally linked with cancer of the nasal cavity (Fukuda et al., 1988, 1990), cervix (Slattery et al., 1989) and bladder (IARC, 1986). The United States Environmental Protection Agency has classified ETS in Group A: Human carcinogen (U.S. EPA, 1992b). The International Agency for Research on Cancer has classified *tobacco smoke* in Group 1: Human carcinogen (IARC, 1986).

The State of California under Proposition 65 has determined that *tobacco smoke* is a carcinogen (CCR, 1996). The preliminary recommended potency value of ETS for use in cancer risk assessments is 2.8×10^{-5} (micrograms per cubic meter)$^{-1}$. In other words, the potential excess cancer risk for a person exposed over a lifetime to 1 $\mu g/m^3$ of ETS is estimated to be no greater than 28 in 1 million (CAPCOA, 1993).

Table I
Common Constituents of Environmental Tobacco Smoke

(Synonyms: passive smoke; second-hand smoke)

Acetaldehyde	Cholesterol	N-Nitrosodiethanolamine
Acetic acid	Dimethylamine	N-Nitrosodiethylamine
Acetone	Formaldehyde	N-Nitrosodimethylamine
Acrolein	Formic acid	N´-Nitrosonornicotine
4-Aminobiphenol	Glycolic acid	N-Nitrosopyrrolidine
Ammonia	Harman	NNK
Anatabine	Hydrazine	o, m, & p-Cresol
Aniline	Hydrogen cyanide	Particulate matter
Benzene	Hydroquinone	Phenol
Benz[a]anthracene	Isoprene	Polonium-210
Benzo[a]pryrene	Lactic acid	Propionaldehyde
Benzoic acid	Methylamine	Pyridine
γ-Butyrolactone	Methyl chloride	Quinoline
Cadmium	3-Methylpyridine	Succinic acid
Carbon dioxide	2-Naphthylamine	Toluene
Carbon monoxide	Nickel	2-Toluidine
Carbonyl sulfide	Nicotine	3-Vinylpyridine
Catechol	Nitrogen oxides	Zinc

(NRC, 1986b)

Table II

Environmental Tobacco Smoke Emission Factors*

Compound	Emission Factor (ng/mg)		Emission Factor (μg/cig.)	
	Average± Std. Dev.[a]	1R4F[b]	Average± Std. Dev.[a]	1R4F[b]
Acetaldehyde	3,340±525	3,430	2,150±477	2,220
Acrolein[c]	(126±109)	(120)	(86±86)	(78)
Acrylonitrile	154±16	185	99±18	120
Benzene	630±31	653	406±71	423
1,3-Butadiene	236±29	276	152±27	179
2-Butanone (MEK)	451±25	585	291±56	379
Butyl acetate[d]	<4	<4	<3	<3
Butyaldehyde[d]	<29	<29	<18	<18
m,p-Cresol[e]	128±27	106	83±26	68
o-Cresol[e]	55±11	59	35±5	38
Ethyl acetate[d]	<6	<6	<4	<4
Ethyl acrylate[d]	<5	<5	<3	<3
Ethylbenzene	157±14	178	130±10	89
Formaldehyde	2,040±414	2,060	1,310±348	1,330
3-Methyl-1-butanol[d]	<23	<23	<14	<14
Nicotine	1,410±260	1,540	919±214	993
N-Nitrosodiethylamine[d]	<0.033	<0.033	<0.020	<0.020
N-Nitrosodimethylamine	0.88±0.11	0.69	0.57±0.12	0.44
N-Nitrosomorpholine[d]	<0.033	<0.033	<0.020	<0.020
N-Nitrosopyrrolidine	0.16±.02	0.15	0.10±0.02	0.10
Phenol[e]	438±76	368	281±61	238
Pyridine	663±126	989	428±122	641
Pyrrole	626±94	816	402±90	529
Styrene	229±16	250	147±24	162
Toluene	1,020±78	1,130	656±107	732
3-Vinylpyridine[e]	1,020±149	1,054	662±155	683
m,p-Xylene	467±40	504	299±52	327
o-Xylene	104±13	115	67±16	75
$PM_{2.5}$[e]	12,400±1,300	11,900	8,100±2,000	7,700

* (Daisey et al., 1994)

a. Average ± Standard Deviation for six commercial cigarettes.

b. Kentucky reference cigarette which reflects current U.S. market shares of various cigarettes.

c. Use of acrolein emission factors for exposure modeling is not recommended.

d. Less-than values are lower limits of detection.

e. Emission factors are corrected for deposition losses to chamber surfaces.